Brit. Heart J., 1965, **27**, 684.

RENAL RESPONSE TO PAROXYSMAL TACHYCARDIA

BY

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Received August 10, 1964

The pattern of water and solute excretion during paroxysmal tachycardia has been studied by Borst and his colleagues (Blomhert, 1951; Borst, 1941; Borst *et al.*, 1960). They showed that with the onset of paroxysm there was a striking water diuresis which continued for the duration of the paroxysm and then stopped. An increase in sodium chloride excretion was also recorded at some time in the attack. Creatinine excretion was constant. A case studied by Wood (1963) developed a diuresis during a paroxysm which was associated with an increased output of sodium. This was not a water diuresis as the urine specific gravity was high.

Wood (1963) accumulated statistics on the diuresis accompanying paroxysmal tachycardia. Diuresis occurs with all forms of paroxysmal arrhythmia when the heart rate exceeds 110 a minute; and with heart rates varying from 120 to 280 a minute. Diuresis starts after 20 to 30 minutes, but sometimes not for 60 minutes after the onset, the time interval being remarkably constant in the individual: it does not occur with a paroxysm of 5 to 20 minutes' duration.

Because these changes are of interest and of possible physiological importance, the changes in urine composition were recorded during two episodes of paroxysmal tachycardia in a young woman.

Case Report

A healthy, single girl of 23 was investigated. She had experienced bouts of palpitations lasting a few minutes from the age of 16 years. For 2 years the episodes had become more frequent and tended to last several hours, and she had noted that occasionally she passed more urine during attacks. Physical examination between attacks was normal. Pulse was regular with a rate of 68 a minute; blood pressure was 120/70 mm. Hg; electrocardiogram showed sinus rhythm with a rate of 70 a minute. During attacks, apex rate rose to 160 a minute. Venous pressure was not raised at any time. Blood pressure rose to 130/100 mm. Hg at the beginning of some attacks. Electrocardiograms during paroxysms showed a supraventricular nodal tachycardia. Carotid sinus pressure was without effect.

Investigations. Plasma sodium, 137 mEq/l.; potassium, 4 mEq/l.; chlorides, 104 mEq/l.; bicarbonate, 24·8 mEq/l.; osmolality, 295 m.osm./kg.; urea, 32 mg./100 ml.; true creatinine, 0·7 mg./100 ml.; 24-hour endogenous creatinine clearance, 132 ml./min.; random 24-hour urine collection, 1870 ml., with excretion of sodium, 56 μEq/min., potassium, 73 μEq/min., and urea, 300 μM./min. Hæmoglobin, 13·5 g./100 ml. Chest radiograph was normal. Urine contained no albumin and was sterile on culture.

At the beginning of the paroxysm, the subject was put to bed in a recumbent position. Free collections of urine were timed. During the paroxysm, venous blood was withdrawn into heparinized tubes and centrifuged. Blood pressure was recorded every 15 minutes with a mercurial sphygmomanometer. Apex rate was recorded every 15 minutes. Only small quantities of fluid were ingested during the paroxysm. Observations were continued for several hours after the heart rate fell below 100 a minute.

Biochemical measurements on plasma and urine consisted of osmolality (Fiske osmometer), sodium and potassium (flame photometer), urea (Nesslerization), and creatinine (Jaffe reaction in urine, true creatinine in plasma).

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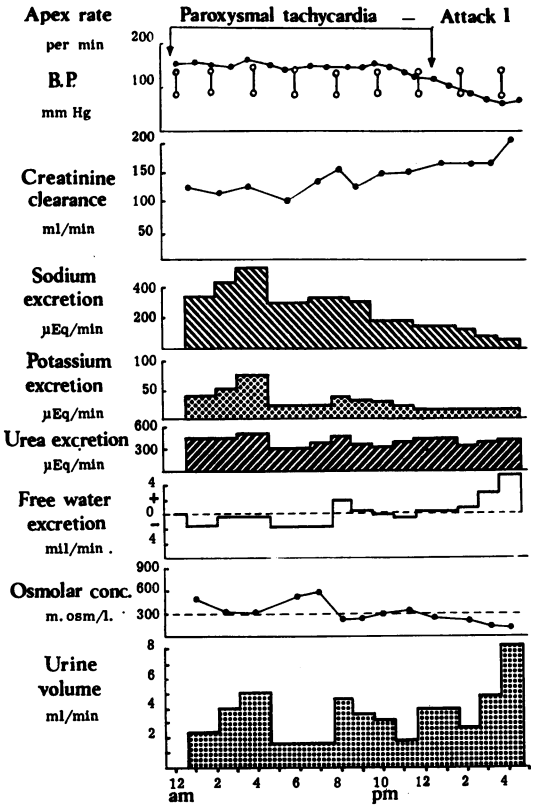

FIG. 1.--First observed episode of paroxysmal tachycardia in the patient.

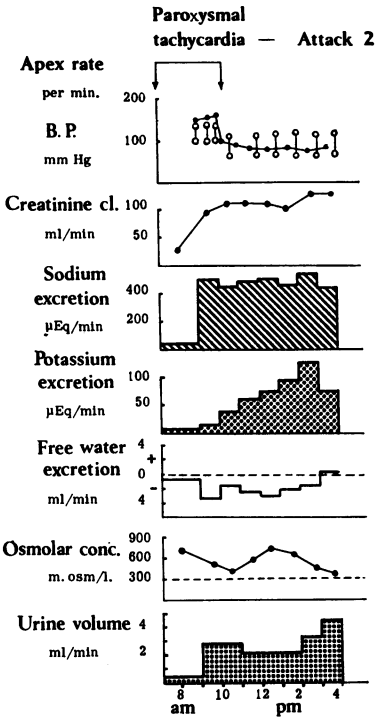

FIG. 2.---Second attack of paroxysmal tachycardia.

RESULTS

The biochemical composition of the urine is illustrated in Fig. 1 and 2. Heart rate remained about 160 a minute in both paroxysms. Urine flow increased during and after both episodes of paroxysmal tachycardia. Early in the first attack increased urine flow was associated with a significant rise in sodium excretion. There was a slight increase in potassium excretion. Towards the end of the paroxysm, and afterwards, there was a striking rise in urine flow with increased free water excretion.

In the second attack, the increase in urine flow was associated with a raised sodium excretion. Potassium excretion was slightly raised initially. Free water excretion was negative. Creatinine and osmolar clearances were considerably depressed for the first two hours.

Figure 3 shows the relationship between osmolar clearance and urine flow in both episodes of paroxysmal tachycardia. This graph illustrates that, if the urine were isotonic with plasma, osmolar clearance would equal urine flow and all points would fall on the diagonal line. Displacement of points to the left of this line indicates reabsorption of solute-free water from isotonic glomerular filtrate. The amount of water reabsorbed is proportional to the horizontal distance from the diagonal. Displacement to the right of this line is proportional to the amount of free water excreted. It may be seen that in the first attack water reabsorption was present early in the attack and free water excretion occurred after the attack. The second attack was accompanied by water reabsorption only. The raised urine volume present in the early stages of both attacks was due to increased osmolar clearance.

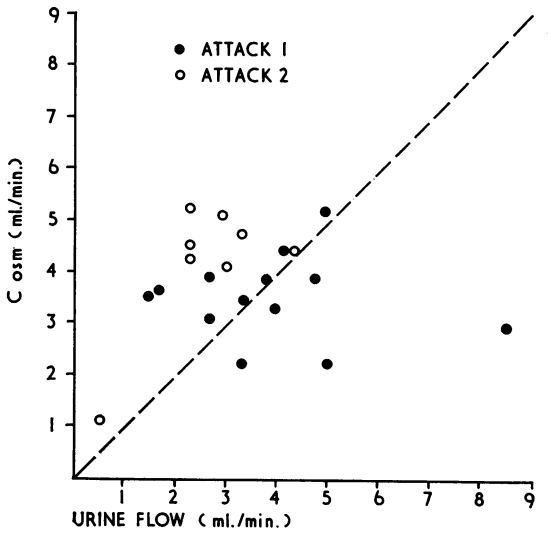

FIG. 3.—Urine flow plotted against osmolar clearance in the two episodes in the patient. Urine water is divided into two fractions according to the following formulae: (1) $Cosm = \frac{Uosm}{Posm} \times V$ (2) $C_{H_2O} = V - Cosm$ (3) $T^c_{H_2O} = Cosm - V$

V represents urine flow (ml./min.) and Uosm and Posm represents the osmolal concentrations of urine and plasma respectively. The first fraction is the osmolar clearance Cosm, which is calculated according to Formula (1), and represents the volume of urine water which contains the urine solutes in isotonic concentration with plasma. The second fraction represents the net excess or deficit of water in the urine after subtracting osmolar clearance. This is the free water clearance and is calculated from Formula (2). The expression $T^c_{H_2O}$ indicates water reabsorption, or negative free water clearance, and is calculated according to Formula (3). These fractions are mathematical and do not describe discrete functional operations within the kidney, but they serve to quantitate tubular concentrating and diluting ability.

DISCUSSION

Urine flow is variable during an episode of paroxysmal tachycardia. Borst (1941) noted oliguria early in the attack and this was demonstrated in the second attack. When polyuria occurs it may be associated with a raised osmolar clearance, due to increased sodium excretion, or a raised free water excretion. The diuresis of sodium or water does not occur at any set time, and may take place at any time during the paroxysm. The present data show that a water diuresis can occur after the paroxysm, in accord with clinical observations on urine flow by Friedberg (1956) and Katz and Pick (1956). Oliguria in this patient occurred when glomerular filtration rate was considerably depressed. Polyuria occurred when glomerular filtration was not depressed, and must have resulted from altered function of the renal tubule, namely decreased tubular reabsorption of sodium or water. The mechanism of these changes is not known.

Borst was impressed with the similarity of the urine changes obtained in paroxysmal tachycardia and after infusion of saline into normal subjects. A water diuresis was obtained by expanding plasma volume (Blomhert *et al.*, 1951; Strauss *et al.*, 1951). Borst proposed the hypothesis that the water diuresis of paroxysmal tachycardia resulted from stimulation of the mechanism controlling extracellular fluid volume. Gauer, Henry, and Sieker (1961) reviewed the problem of volume control and stressed the role of volume receptors in the cardiac atria. Hæmodynamic changes during attacks of paroxysmal tachycardia have been obtained by Ferrer *et al.* (1949) and Saunders and Ord (1962). These changes, which commence with onset of the paroxysm and stop when it ceases, consist of a steep rise in pressure within both atria, with the development of large atrial pulse waves. Little change in arterial pressure and cardiac output occurred. Sodium diuresis occurs after hypotonic expansion of extracellular fluid volume (Leaf *et al.*, 1953; Strauss *et al.*, 1952). The mechanism of this sodium diuresis is not clear, but Jones, Barraclough, and Mills (1963) have shown that aldosterone is not responsible. The variable excretion of sodium and water observed in this patient did not appear to be related to the stimulus of atrial pressure waves which may have been present during the paroxysm.

The abrupt onset of palpitations, which may continue unabated for many hours, produces anxiety and apprehension. The emotional tension may be an important factor in the production of diuresis. Hinkle, Edwards, and Wolf (1951) have shown that apprehension can initiate a water diuresis, and Miles and de Wardener (1953) demonstrated a chloride diuresis from emotion. The variable time of onset of either a water or a salt diuresis, occurring sometimes during and

sometimes after the episode of paroxysmal tachycardia, tends to support an emotional ætiology of the diuresis.

SUMMARY

The diuresis associated with paroxysmal tachycardia may be a water or a salt diuresis. It may occur during or after the paroxysm. The mechanism of production is not known. Renal tubular factors influencing the handling of water and sodium are important. The possible significance of the mechanism controlling volume regulation and emotional factors in the production of diuresis is discussed.

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